optimal management of blood pressure and full renin–angiotensin blockade. But since we are not satisfied their benefit is proven even in that setting, we do not include corticosteroids in our main treatment recommendations in the table or abstract of our article. What Dr Locatelli *et al.* regard as 'nihilism', we regard as pragmatic realism avoiding a treatment regimen with some toxicity when evidence in favor of its use is insecure.

Unfortunately, these uncertainties will not easily be resolved. Modern management with adequate blood pressure control and renin–angiotensin blockade is reducing proteinuria and slowing the progression of a significant proportion of patients with IgA nephropathy. Proof that treatments additional to this regimen are effective will therefore require larger and more prolonged treatment trials unless surrogate markers of progression become more discriminatory, and our ability to define risk of progression in individual patients becomes more refined.

1. Locatelli *et al.* Steroids and IgA nephritis. *Kidney Int* 2006, in press.

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HIVAN is increasingly less common in HIV-positive Black Africans living in Europe

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To the Editor: Gerntholtz *et al.*[1] report their series of kidney biopsies from Black African human immunodeficiency virus (HIV)-positive patients, adding to a growing literature suggesting HIV-associated nephropathy (HIVAN) to be less common than originally described.[2,3] They found HIV immune complex kidney disease to be as common as HIVAN, and describe for the first time a 'ball-in-cup' appearance on silver-methenamine staining or electron microscopy caused by subepithelial deposits associated with basement membrane reaction, placing this lesion as an intermediate between classical post-infectious and membranous glomerulonephritis.

On review of 10 Black African HIV-positive patients biopsied in East London after July 2004, we found a similar prevalence of HIVAN (3/10) and immune complex-mediated glomerulonephritis with mesangial matrix increase and crescent formation (3/10). The 'ball-in-cup' lesion newly described as typical of HIV immune complex kidney disease was apparent in only one of the three such cases on re-examining renal tissue. Our patients were similar in age (30–45 years), renal function (creatinine $462 \pm 620\ \mu$mol/l, mean $\pm$ s.d.), serum albumin (28 ± 9 g/l), and CD4 count (210 ± 155 copies/ml). HIV viral load was $4.28 \pm 0.88 \times 10^6$/l, and none were intravenous drug users or hepatitis B positive.

Unlike the Gerntholtz series in which of those known to follow-up 58% died, the outcome was generally good (with universally good response to anti-retroviral drugs). Despite often advanced presentation, only one patient has died to date after a mean 46 weeks follow-up, and of two patients requiring hemodialysis (both with HIVAN), one has since recovered independent renal function. In Black African migrants living in London, similar rates of HIVAN and immune complex-mediated kidney disease are seen to that described in Johannesburg – the renal and patient outcome is however very different, emphasizing the importance of readily available anti-retroviral therapy.

1. Gerntholtz TE, Goetsch SJ, Katz I. HIV-related nephropathy: a South African perspective. *Kidney Int* 2006; **69**: 1885–1891.
2. Glassock RJ, Cohen AH, Danovitch G *et al.* Human immunodeficiency virus (HIV) infection and the kidney. *Ann Intern Med* 1990; **112**: 35–49.
3. Szczech LA, Gupta SK, Habash R *et al.* The clinical epidemiology and course of the spectrum of renal diseases associated with HIV infection. *Kidney Int* 2004; **66**: 1145–1152.

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Response to 'HIVAN is increasingly less common in HIV-positive Black Africans living in Europe'

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The kidney biopsy experience of Cove-Smith *et al.*[1] in an East London setting with Black African human immunodeficiency virus (HIV)-positive patients is interesting in its parallels to ours. We feel that this emphasizes the fact that not all that is kidney disease in the context of HIV infection is classic HIV-associated nephropathy. There are many other forms of renal pathology which occur, emphasizing the need for renal biopsy to allow full clarification.

Although detailed histopathological clarification does not carry proven therapeutic implications at this stage, we are at present treating all those with HIV-associated nephropathy and HIV immune complex kidney disease with angiotensin-converting enzyme inhibitors and antiretroviral therapy, regardless of the CD4 count, as we feel that the virus is involved in the pathophysiology. This contrasts with our published experience,[2] when antiretroviral therapies were not used as they were not available to